

# MICROCOMPUTER ANALYSIS OF O<sub>2</sub> TRANSPORT AND TISSUE PO<sub>2</sub> IN SHOCK†

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## INTRODUCTION

Oxygen is the ultimate oxidizing agent and is essential in the body to accept electrons which are produced by metabolic reactions. Without O<sub>2</sub> the tissues and ultimately the body would die. Oxygen delivery to the body depends on two different physical principles: bulk flow and diffusion. These mechanisms alternate in their roles in transporting O<sub>2</sub> to the tissues. Pressure gradients, caused by the respiratory muscles, bring O<sub>2</sub> containing gases from the environment to the alveoli, where diffusion then moves O<sub>2</sub> down its diffusion gradient from the alveoli into the pulmonary capillaries. Bulk flow of blood transports the oxygenated blood to the tissues; diffusion again moves the O<sub>2</sub> from the blood into the cells where O<sub>2</sub> is utilized within the mitochondria, the so-called "metabolic furnaces"[1].

O<sub>2</sub> delivery can be reduced by a dysfunction anywhere along the O<sub>2</sub> cascade from the ambient atmosphere to the mitochondria. Complete discussion of O<sub>2</sub> transport must include consideration of all of the factors comprising the O<sub>2</sub> cascade. However, this paper is concerned only with the factors affected by reductions in blood flow. Recent monographs on O<sub>2</sub> transport can be consulted for discussion of the remaining factors.

The present program consists of two main subroutines: Blood Oxygen Content and Tissue Oxygen Tension. The pulmonary uptake of O<sub>2</sub> can be studied using either a three compartment model or ventilation/perfusion ( $V/Q$ ) relationships. The  $V/Q$  subroutine uses multiple or single mode  $V/Q$  distributions to determine gas exchange using a log-normal population of alveoli. Additional variables can be adjusted to determine the arterial and mixed-venous blood gas composition, and these values are then fed into the second subroutine, which allows analysis of various factors which determine tissue O<sub>2</sub> tension. The Tissue Oxygen Tension subroutine is also subdivided: a modified Krogh–Erlang model[2], which provides a three-dimensional plot of capillary and tissue O<sub>2</sub> tensions; and a Piiper model[3] which includes the effect of diffusion shunt and treats the tissue as a well stirred compartment. However, minimal and maximal tissue O<sub>2</sub> tensions are calculated while using the Piiper model, since the intercapillary distance is allowed to vary depending on the O<sub>2</sub> delivery by diffusion. A diffusion–perfusion ratio is calculated (Haab[4]) to distinguish between diffusion or perfusion limited conditions in the tissues. The Tissue O<sub>2</sub> Tension subroutine requires the input of O<sub>2</sub> consumption and blood flow for the particular organ or tissue, as well as the capillary density and the amount of perfusion shunt, if any, which is present. The results provide estimates of blood and tissue O<sub>2</sub> tensions, diffusion/perfusion coefficients[4], amount of O<sub>2</sub> delivered and the size of the active capillary bed.

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† Supporting diskette available.

## BACKGROUND OF ANALYSIS

The work of Wagner and coworkers[5, 6] has shown that the population of  $V/Q$  ratios can usually be characterized by a log-normal distribution. This program characterizes the population of  $V/Q$  ratios by three parameters: the mean  $V/Q$  ratio, the standard deviation of the population and the proportion of total blood flow going to that mode of the distribution. The calculations then provide estimates of alveolar  $PO_2$  ( $PALVO_2$ ), alveolar  $PCO_2$  ( $PACO_2$ ),  $O_2$  uptake and alveolar ventilation for seven points in the distribution. Multiple modes of the  $V/Q$  distribution can also be defined, which makes it possible to study the effects of various aspects of  $V/Q$  on the gas exchange properties of the system.

Conversion between  $O_2$  content and  $PO_2$  is performed by using a regression analysis of a standard dissociation curve using data from Altman and Dittmer[7] and Severinghaus[8]. Regression analysis was carried out using a curve fitting program, termed Curfit, (Spain[9]), that provides a formula that is a version of the linearized Hill equation. This regression analysis yielded a correlation coefficient of 0.9940, standard error of estimate was 1.94 and an F value of 11401 for data ranging between  $PO_2$  of 1 and 700 mm Hg for hemoglobin saturations of 1%–99.95%. The calculation of blood  $PO_2$  is performed by calculating a standard  $PO_2$  using the regression constants which were derived and then adjusting the  $P_{50}$  of hemoglobin due to changes in pH,  $PCO_2$  and temperature by use of the Kelman algorithm[10].

The Fick Principle is essential when analyzing the relationship between blood flow and  $O_2$  content in the arterial and venous blood. The Fick Principle is an expression of the conversion of mass and can be written

$$VO_2 = QT(CAO_2 - CVO_2),$$

where  $VO_2$  is the oxygen consumption, QT is blood flow,  $CAO_2$  is the arterial  $O_2$  content,  $CVO_2$  is the venous  $O_2$  content.

If total body  $O_2$  consumption is considered, then QT represents cardiac output, and  $CVO_2$  is the mixed venous blood which should be sampled from the pulmonary artery. The Fick relationship holds true for each organ as well so that  $VO_2$  for any organ can be calculated from the organ blood flow and the respective arterial and venous  $O_2$  contents.

Rearranging the above equation gives

$$CVO_2 = CAO_2 - VO_2/QT.$$

This relationship has been used for years to evaluate the  $O_2$  delivery system for the entire body, since if  $O_2$  consumption rises or blood flow decreases, then  $PVO_2$  decreases. When  $PVO_2$  falls below a critical level this generally indicates that  $O_2$  delivery is inadequate and that tissue anoxia is present (Mithoeffer *et al.*[11]). The intracapillary  $PO_2$  is important, since as it falls, the pressure head causing diffusion to occur is lost. Thus, even though blood flow may be the primary factor in limiting  $O_2$  delivery, it eventually leads to an inadequate capillary  $O_2$  tension so that diffusion limitation may also play a role in producing anoxic or hypoxic conditions. It is important to differentiate between conditions leading to anoxia due to inadequate blood flow or to diffusion limitation since each one requires a different treatment. Anoxia due to inadequate perfusion may be treated with vasodilators, cardiac stimulants or volume infusion, whereas diffusion impairment may be corrected by supplemental  $O_2$  administration. A model developed by Haab[4] is used in the present program to calculate a diffusion/perfusion ratio which provides an estimate

as to whether O<sub>2</sub> delivery is limited by diffusion or perfusion conditions. Three estimates of tissue PO<sub>2</sub> are provided by the program: An average PO<sub>2</sub> is calculated from the Krogh-Erlang model as well as minimal and maximal PO<sub>2</sub> as calculated from the Piiper model. These latter two values arise, since the intercapillary distance is allowed to vary depending on the capillary PO<sub>2</sub> and the rate of O<sub>2</sub> consumption.

## DESCRIPTION OF THE PROGRAM

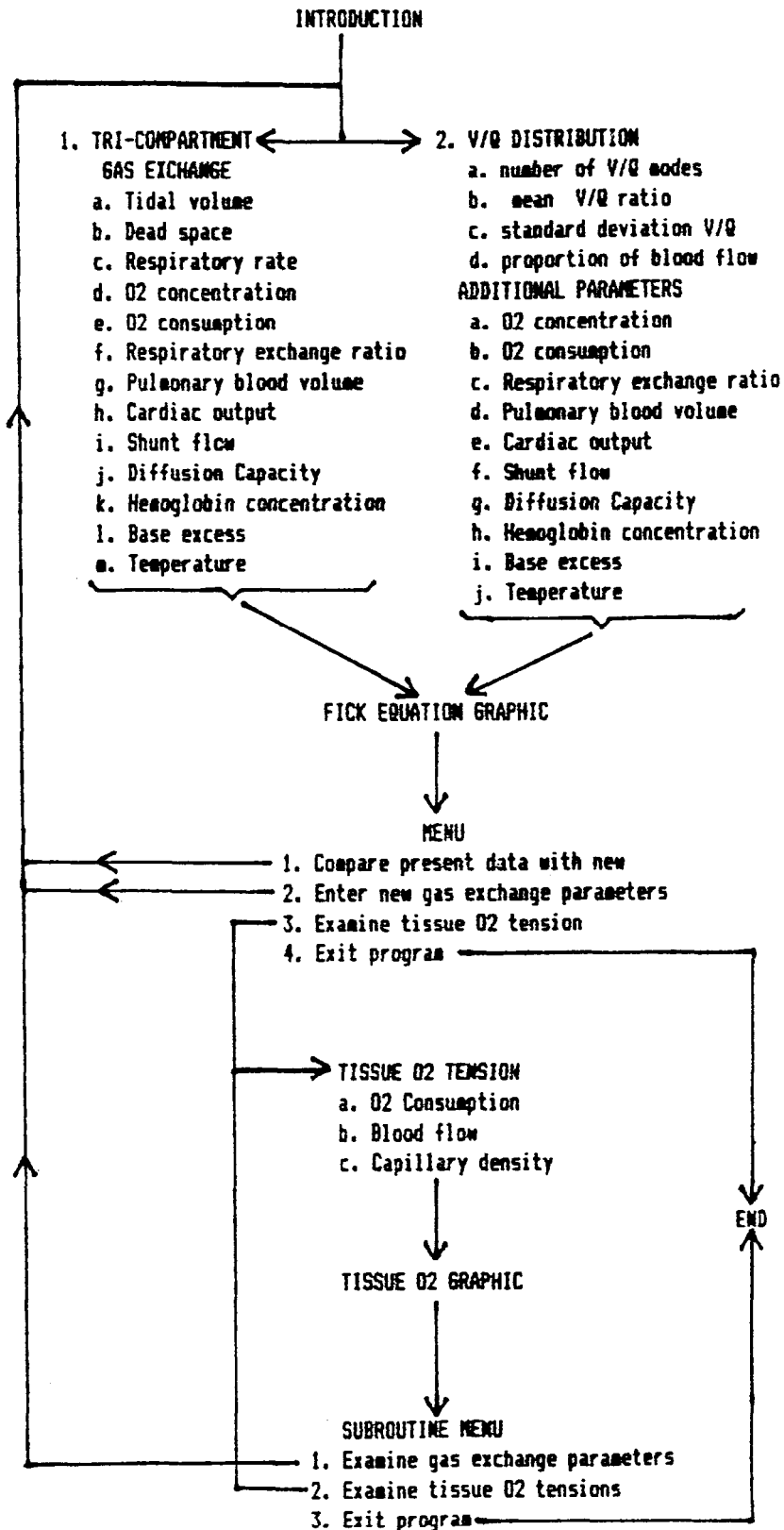
The program is written in BASICA and is listed in the appendix. Table 1 shows an outline of the program with the various options that are available. The program is 19,000 bytes in length and runs on any MS-DOS microcomputer (IBM-PC or compatible) containing at least 128K of RAM, 1 disk drive and graphics capability. The program utilizes standard graphics commands from BASICA and, therefore, will run on any 16-bit microcomputer utilizing the appropriate version of BASICA for that machine.

Lines 10–420 dimension the variables and provide a brief introduction to the program, which is completely menu driven. Lines 440–470 provide a branch point so that the user can select either a three compartment or  $V/Q$  analysis of gas exchange. Line 480 lists the default values for 16 variables used in the gas exchange algorithm. Lines 520–700 provide the input to define the  $V/Q$  characteristics, while lines 720–840 list additional variables that can alter gas transport. Lines 900–1380 provide input for the three compartment analysis of gas exchange and transport. Lines 1390–1470 provide the second menu of choices which branch back to the gas exchange algorithms, tissue O<sub>2</sub> tension or exit the program. The data from the latest ABG determinations are fed into the Tissue O<sub>2</sub> Tension subroutine if that choice is selected.

Lines 1500–1650 draw a graphic of the data from the Fick equation which is used to display the blood gas relationships[12]. The actual calculations of gas exchange and blood gas parameters begin in line 1690 and continue through line 2310. Variables have been named according to the accepted nomenclature as much as possible within the constraints of the BASIC language, in order to make the program as transparent as possible. Several important exceptions to the usual abbreviations are ALV = alveolar, CAP = capillary; CAO<sub>2</sub> = arterial O<sub>2</sub> content, etc. Lines 1810–1880 calculate the fractional value for blood flow and alveolar ventilation based on the log-normal distribution and the calculated  $V/Q$  ratio (line 1800). The CAO<sub>2</sub> is calculated at different points of the vascular system in lines 1690, 2040, 2190 and 2250 for mixed venous, pulmonary capillary, arterial and venous blood, respectively. After determining the O<sub>2</sub> content of the blood a gosub to the routine at lines 3190–3250 is performed to convert the content to a standard PO<sub>2</sub> and then to adjust the PO<sub>2</sub> using the Kelman algorithm[10]. The diffusion properties of the alveolar membrane depend on both pulmonary blood volume and cardiac output which determine the transit time of the red cells in the pulmonary capillaries as well as the diffusion capacity of the lungs for O<sub>2</sub> (DLO<sub>2</sub>). The pulmonary diffusion of O<sub>2</sub> is modeled using an iteration of the blood O<sub>2</sub> content during 50 intervals of the red cell transit time (2020–2090).

Line 2320 begins the Tissue O<sub>2</sub> subroutine which requires the input of four parameters for the particular organ being studied (2350–2380). Lines 2550–2630 draw the outline of the three-dimensional capillary segment, while lines 2640–2770 are used to plot out the tissue PO<sub>2</sub> profiles which are calculated at 2780–3170. Tissue O<sub>2</sub> tension is calculated in steps since the intercapillary distance [RT(L%)] is allowed to vary depending on the PO<sub>2</sub> which is present in order to drive the O<sub>2</sub> diffusion to match the tissue O<sub>2</sub> consumption (VO<sub>2</sub>). The capillary O<sub>2</sub> content [CCAPI(1%)] is assumed to decrease linearly down the capillary (line 2850).

Table 1. Program outline



## DISCUSSION

The arterial O<sub>2</sub> content is a function of the diffusion properties of the lungs, the hemoglobin concentration, the alveolar ventilation, the P<sub>50</sub> of hemoglobin, the cardiac output, the amount of true shunt ( $V/Q = 0$  or blood flow not exposed to alveoli), the  $V/Q$  ratio and the venous O<sub>2</sub> content. All of these factors are incorporated into this model so that it should be possible to simulate virtually any condition and observe its effect on gas exchange. The first seven of these factors are well understood and will not be discussed further. The last factor has been shown to be extremely important in determining the arterial O<sub>2</sub> content[13] and is frequently overlooked as a cause of arterial hypoxia. The effect of venous O<sub>2</sub> levels on arterial O<sub>2</sub> content is best described by rearranging the shunt equation:

$$QS/QT = (C_{IO_2} - C_{AO_2}) / (C_{IO_2} - C_{VO_2}),$$

where

QS is the shunt flow,

C<sub>IO<sub>2</sub></sub> is the pulmonary capillary O<sub>2</sub> content.

Thus  $CAO_2 = C_{IO_2} - [QS/QT \cdot (C_{IO_2} - C_{VO_2})]$ .

It can be seen that increases in shunt flow or decreases in QT and CVO<sub>2</sub> will reduce the arterial O<sub>2</sub> content. Therefore, the reduction in cardiac output has a powerful effect on O<sub>2</sub> transport, since the O<sub>2</sub> transport is reduced by the decreased cardiac output, and CAO<sub>2</sub> is further reduced by the lowering of the CVO<sub>2</sub>, which accompanies any decrease in the cardiac output. This effect can be well documented by the use of this program (Fig. 1).

The coefficient of oxygen delivery ( $COD = QT \cdot CAO_2 / VO_2$ ) was proposed by Mithoeffer *et al.*[11] to evaluate the O<sub>2</sub> delivery system. However, it has been shown that the

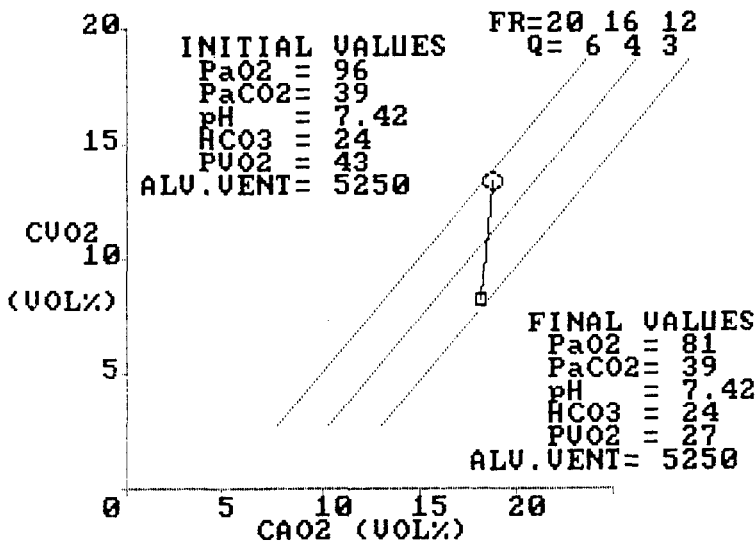


Fig. 1. A graphic analysis of the Fick Principle. Abscissa is arterial O<sub>2</sub> content; ordinate is venous O<sub>2</sub> content. The diagonal lines represent isoflow conditions. The diagram is constructed such that if O<sub>2</sub> consumption (VO<sub>2</sub>) is 300 ml/min the lines then represent cardiac output (QT). If VO<sub>2</sub> is not 300 ml/min then the diagonal lines represent a flow ratio which equals QT/VO<sub>2</sub>. The circle indicates the initial conditions, whereas the square represents the coordinate following the intervention. This diagram indicates the changes that occur with a reduction in cardiac output from 5.5 to 3 l/min. Note the decrease in CAO<sub>2</sub> due to the decrease in cardiac output.

COD can increase, decrease or remain constant under various conditions when  $O_2$  transport is limited[14]. The factors which determine the  $O_2$  delivery can be better evaluated by graphing the Fick equation[12]. If  $CAO_2$  is plotted on the abscissa and  $CVO_2$  on the ordinate, then decreases in the cardiac output cause the coordinate to move downward and to the left; i.e. to lower arterial and venous  $O_2$  contents (Fig. 1). In contrast, if  $O_2$  delivery is reduced because of a decrease in hemoglobin concentration or  $O_2$  tension then the coordinate moves diagonally, parallel to the flow lines (Fig. 2). Thus the factors affecting the  $O_2$  delivery, either changes in  $O_2$  content or the bulk flow of blood, can be evaluated by this type of graphic analysis, and the effects of treatment or progression of the disease process can be followed.

Measurements of  $PVO_2$  or  $CVO_2$  can provide evidence regarding the overall efficiency of the  $O_2$  delivery system. However, each organ has its own unique ratio of  $O_2$  consumption and blood flow which generates a unique value for the respective venous  $O_2$  tension and content. Therefore, it is possible that the overall  $O_2$  delivery system is adequate so that mixed venous  $PO_2$  is normal while blood flow is inadequate to a specific organ.

The actual tissue  $PO_2$  depends not only on the ratio of  $O_2$  consumption and blood flow but is also dependent on the microvascular geometry, the degree of  $a-v$  shunting, the counter-current mechanism between the arterioles and venules and especially on the number of perfused microvessels, which is the primary determinant of the intercapillary distance. None of these parameters can be measured clinically, but examination of their effects by means of a mathematical model provides insight into their interaction. The  $PO_2$  decreases in an exponential function as  $O_2$  diffuses from the capillary into the tissues even though  $O_2$  content decreases linearly down the capillary[1]. The  $PO_2$  approaches zero in the mitochondria, where  $O_2$  is consumed, even when  $O_2$  is adequately supplied.

The capillary  $PO_2$  ( $PCAO_2$ ) determines the driving force for diffusion into the tissues. The  $PCAO_2$  is determined by the hemoglobin concentration, the  $P_{50}$ , the amount of

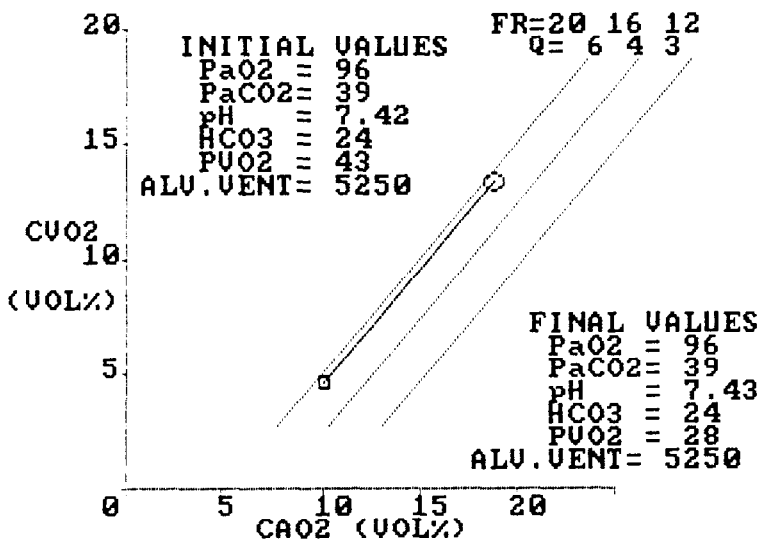


Fig. 2. The effect of changes in  $O_2$  transport due to changes in the  $O_2$  content as depicted on plot of the Fick equation. Symbols as in Fig. 1. The diagram indicates the changes that are caused only by a change in hemoglobin concentration. Note the movement is parallel to the flow lines since cardiac output was unchanged in the two conditions.

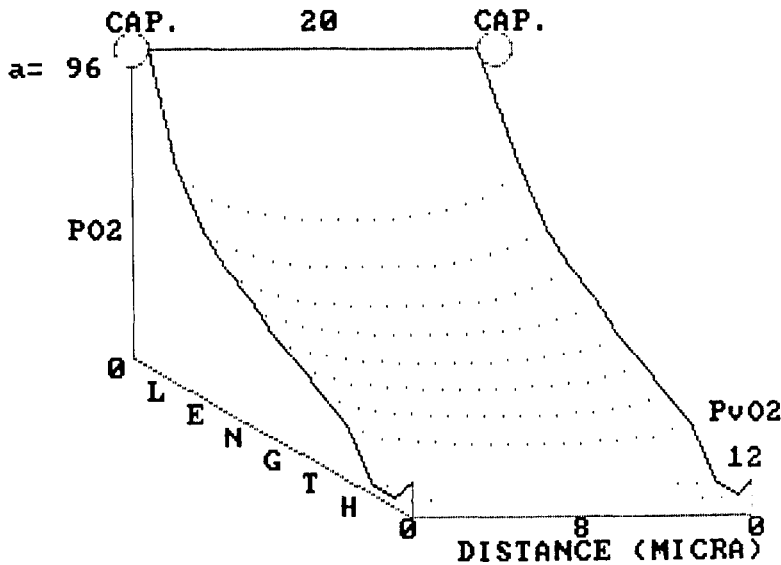


Fig. 3. A three-dimensional plot showing capillary length, intercapillary distance and tissue PO<sub>2</sub>. Note that intercapillary distance varies from the arterial to the venous end of the capillary, which alters the diffusion distance. In this example the diffusion shunt was set at 20%, which caused the minimal capillary O<sub>2</sub> tension to occur proximal to the venous end of the capillary.

blood flow, the distance along the capillary which the blood has traversed and the rate of O<sub>2</sub> utilization by the tissues. It is obvious that predicting the PO<sub>2</sub> in any tissue is a complex problem given the number of variables that are involved. Most of the difficulty arises because of the complex and unknown geometry in the microvasculature. The most common model used for predicting the tissue PO<sub>2</sub> and the presence of tissue hypoxia is the Krogh-Erlang equation. This equation utilizes 15 assumptions in its derivation all of which are probably untrue in most tissues[15]. The most serious errors appear to arise from the assumptions dealing with the microvascular geometry. The Krogh model assumes that all capillaries are parallel, of equal length and receive equal blood flow. Additionally, it is assumed that blood flow is concurrent, i.e. in the same direction, in adjacent capillaries and that there is no exchange between adjacent vessels (shunting). Thus in this model the lowest PO<sub>2</sub> in the capillaries occurs at the venous end, and obviously then venous PO<sub>2</sub> would be a good measure of the minimal vascular PO<sub>2</sub>. However, recent studies indicate that the PVO<sub>2</sub> may not reflect changes in the tissue PO<sub>2</sub> under many different conditions[15, 16].

A modified Krogh model has been developed which incorporates variation in the intercapillary distance along the length of the capillary (Fig. 3). The variable intercapillary distance is based on anatomic evidence and results in a microvascular unit which has been called the Krogh cone rather than the Krogh cylinder by Weibel[1]. The model also allows studying the effects of both diffusion and perfusion shunts on capillary and tissue PO<sub>2</sub>, thus eliminating the assumptions of concurrent flow and no diffusional shunts. Results from this model indicate that mean tissue PO<sub>2</sub> and venous PO<sub>2</sub> can be dissociated whenever there is a-v shunting, if the number of perfused capillaries is reduced, and if counter-current flow exists in adjacent capillaries. Thus PVO<sub>2</sub> is not an accurate measure of tissue O<sub>2</sub> tension under all conditions. Accurate evaluation of the O<sub>2</sub> delivery system is obviously an important factor in treating cases of shock. While PVO<sub>2</sub> has been used in the past as an indication of tissue anoxia, it is now recognized as having marked limitations as noted above and as emphasized recently by Miller[16].

Newer techniques of evaluating  $O_2$  delivery to the tissues include measuring tissue  $O_2$  tension using invasive methods[17], transdermal  $O_2$  and  $CO_2$  tensions[18] and serum lactate concentrations[19, 20]. Serum lactate appears to be the most promising technique at present, since it can evaluate the delivery of  $O_2$  to the entire body rather than to a few isolated regions as with the other two methods. Several studies have shown that serum lactate concentrations served as a good prognosticator of survival in both animal and human experiments[19, 20]. The present program could be modified to provide lactate production from certain tissues when anaerobic conditions occur and to model lactate uptake by kidney and liver depending on overall flow rates for these tissues when data from animal experiments is available.

This program is available from the author if a blank, soft-sectored disc (IBM compatible) is sent to the author at the following address. Format the disc and indicate if it has been preformatted if your operating system is lower than DOS 2.1. Joseph Boyle, M.D., Dept. Physiology, N.J. Medical School, 100 Bergen St., Newark, NJ 07103.

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## APPENDIX A

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10 *****SHOCK PROGRAM*****
20 *****J.BOYLE, DEPT.PHYSIOLOGY,NJ MED.SCHOOL,NEWARK,NJ 07103*****
30 *****REVISED 5/22/85*****
40 CLS:KEY OFF:WIDTH 80:CLEAR:DEFINT P
50 CLS:DIM PT02(11,21),P1T02(11,21),P2T02(11,21),X1(12),Y1(12),X2(12),Y2(12),DQ(
12),P3T02(12)

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60 PRINT "****O2 Transport and Tissue O2 in Circulatory Shock****"
70 PRINT:PRINT "The venous P02 (PvO2) is widely used as a measure of tissue oxy
genation since"
80 PRINT:PRINT "it supposedly reflects the average tissue P02 under a variety o
f conditions."
90 PRINT:PRINT "However, there are certain instances when PvO2 is dissociated f
rom the"
100 PRINT:PRINT "tissue O2 tension (PtO2). This occurs if there is countercurre
nt flow in cap-"
110 PRINT:PRINT "illaries or if shunting occurs between arterial and venous ves
sels during low "
120 PRINT:PRINT "flow states when capillary perfusion is compromised. In shock
the number of"
130 PRINT:PRINT "perfused capillaries is reduced so that the intercapillary dis
tance is widened."
140 PRINT:PRINT "      PvO2 can be easily determined from the Fick Equation (an
expression of the"
150 PRINT:PRINT "conservation of mass) if the appropriate parameters are known.
"
160 PRINT "          PvO2=f(CaO2-V02/QT)"
170 PRINT "          CaO2=arterial O2 content
180 PRINT "          QT=cardiac output
190 PRINT "          V02=total O2 consumption"
200 GOSUB 3180
210 CLS:PRINT "During shock many physiological parameters are altered so that no
t only cardiac"
220 PRINT:PRINT "output but other factors altering CaO2 are affected. Thus, all
of the factors"
230 PRINT:PRINT "should be examined and their interactions are major factors in
determining the"
240 PRINT:PRINT "determining the total O2 transport and consequently the tissue
O2 tension."
250 PRINT:PRINT "involved in gas transport and can be used to examine the effec
ts of various"
260 PRINT:PRINT "changes on gas transport and tissue O2 tension."
270 PRINT:PRINT "This program consists of two main subroutines:
280 PRINT:PRINT TAB(25)"1. ARTERIAL AND VENOUS BLOOD GASES"
290 PRINT:PRINT TAB(25)"2. TISSUE O2 TENSION (PtO2)"
300 GOSUB 3180
310 PRINT TAB(25)"ARTERIAL AND VENOUS BLOOD GASES"
320 PRINT:PRINT "      Two methods of analyzing the arterial and venous blood ga
ses are provided"
330 PRINT:PRINT "in the program. You may select pulmonary gas exchange using ei
ther a three "
340 PRINT:PRINT "compartment model (Riley and Cournand) or a subroutine using a
distribution of"
350 PRINT:PRINT "ventilation/perfusion ratios (V/Q). The number of modes of V/Q
distribution is"
360 PRINT:PRINT "variable and each mode is assumed to follow a log-normal distr
ibution. The "
370 PRINT:PRINT "amount of true shunt is also variable."
380 PRINT:PRINT TAB(25)"TISSUE O2 TENSION"
390 PRINT:PRINT "      The data from the blood gas subroutine is utilized in thi
s section of the"
400 PRINT:PRINT "program to predict tissue O2 tension. The O2 consumption, bloo
d flow, capillary"
410 PRINT:PRINT "density and degree of a-v shunting must be entered. Graphic di
splay of calcu-"
420 PRINT:PRINT "lated O2 tensions and several estimates of tissue oxygenation
are provided."
430 GOSUB 3180
440 PRINT TAB(20)"PULMONARY GAS EXCHANGE
450 PRINT:PRINT "1. THREE COMPARTMENT ANALYSIS"

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460 PRINT :PRINT "2. V/Q ANALYSIS
470 PRINT :INPUT "ENTER THE NUMBER OF CHOICE THEN PRESS RETURN KEY.";QQ
480 MODE=1:VT=500:VO=150:F=15:PB=760:FIO2=.21:R=.8:VO2=300:VCO2=VO2*R:PBV=500:QT
=5.5:DLO2=32:QS=.25:HGB=15:HCO3=24:TEMP=37:BE=0 'DEFAULT VALUES
490 IF QQ=1 THEN Q3=1:GOTO 900
500 CLS:PRINT TAB(25)"VENTILATION/PERFUSION ANALYSIS"
510 QS=0:Q3=2 'MARKER FOR VQ ANALYSIS
520 LOCATE 2,1:PRINT "DO YOU WISH TO USE ALL NORMAL VALUES FOR THE V/Q DISTRIBUT
ION?"
530 LOCATE 4,1:PRINT "ENTER Y(ES) OR N(O) ":QS=INPUT$(1)
540 IF QS="Y" OR QS="y" THEN N=1:VQ(1)=.95:STDV(1)=.23:QF(1)=1:GOTO 710
550 CLS:LOCATE 2,1:PRINT"*****JUST PRESS ENTER KEY TO RETAIN NORMAL VALUES****
*****"
560 LOCATE 3,1:INPUT ;"1. ENTER THE NUMBER OF MODES OF VQ DISTRIBUTIONS (NORMAL=
1)";N:IF N=VAL(CHR$(13)) THEN N=1
570 IF Q5=1 GOTO 660:REM Q5 USED AS A FLAG DURING CORRECTION SUBROUT.
580 FOR MODE=1 TO N 'DATA INPUT FOR VQ SUBROUTINE
590 LOCATE 4,1:PRINT; "2. ENTER MEAN VQ FOR MODE "MODE " (NORMAL=.95)";:INPUT;VQ
(MODE):IF VQ(MODE) =VAL(CHR$(13)) THEN VQ(MODE)=.95
600 IF Q5=1 GOTO 660:REM PROGRAM CONTROL DURING CORRECTION SUBROUTINE
610 LOCATE 5,1:PRINT; "3. ENTER STANDARD DEVIATION OF VQ FOR MODE "MODE " (NORMA
L=.23)";:INPUT;STDV(MODE):IF STDV(MODE) =VAL(CHR$(13)) THEN STDV(MODE)=.23
620 IF Q5=1 GOTO 660
630 LOCATE 6,1:INPUT ;"4. ENTER FRACTION OF PULMONARY FLOW DISTRIBUTED TO THIS M
ODE (NORMAL=1)";:QF(MODE):IF QF(MODE)=VAL(CHR$(13)) THEN QF(MODE)=1
640 IF Q5=1 GOTO 660
650 QSUM=QSUM+QF(MODE):IF QSUM>1 THEN PRINT"THE SUM OF THE FRACTIONS CANNOT EXCE
ED 1. REENTER DATA.":GOSUB 3180:MODE=1:Q5=0:QSUM=0:GOTO 560
660 LOCATE 10,1:PRINT "ARE ALL DATA ENTERED CORRECTLY? ENTER Y(ES) OR N(O).":QS
=INPUT$(1)
670 IF QS="Y" OR QS="y" THEN CLS:Q5=0:GOTO 700
680 IF QS="N" OR QS="n" THEN LOCATE 15,1:INPUT "ENTER THE NUMBER OF THE INPUT YO
U WISH TO CHANGE, THEN PRESS RETURN KEY";QQ:Q5=1:REM Q5 DIRECTS PROGRAM DIRECTON

690 ON QQ GOTO 560,590,610,630
700 NEXT MODE 'END OF VQ INPUT
710 CLS:WIDTH 80:PRINT "TO CHANGE ANY PARAMETER FROM ITS DEFAULT (NORMAL) VALUE
ENTER THE NUMBER AND"
720 PRINT :PRINT "PRESS <RETURN KEY>. TO START CALCULATIONS PRESS <RETURN KEY> W
ITHOUT ENTERING "
730 PRINT :PRINT "A NUMBER."
740 PRINT "
NORMAL
PRESENT"
750 LOCATE 11,1:PRINT "1. INSPIRED O2 (FIO2) .21",,FIO2
760 PRINT "2. O2 CONSUMPTION (VO2) 300 ml/MIN",,VO2
770 PRINT "3. RQ (VCO2/VO2) .8",,R
780 PRINT "4. PULM.BLD.VOL.(PBV) 500 ml ",,PBV
790 PRINT "5. CARDIAC OUTPUT (QT) 5.5 L/MIN.",,QT
800 PRINT "6. DIFFUSION O2(DLO2) 32 ML/MIN/mm Hg",,DLO2
810 PRINT "7. SHUNT FLOW (QS) 0 L/MIN",,QS
820 PRINT "8. HEMOGLOBIN (HGB) 15 GM/DL",,HGB
830 PRINT "9. BASE EXCESS 0 MEQ/L",,BE
840 PRINT "10. TEMPERATURE (TEMP) 37 DEG C",,TEMP
850 LOCATE 24,1:PRINT "
";
855 IF R>1 OR R<.7 OR TEMP>42 OR TEMP<32 OR ABS(BE)>15 OR HGB>22 GOTO 3260
860 LOCATE 24,1:INPUT;QQ
870 IF QQ=0 THEN GOSUB 1660:GOSUB 1500:GOSUB 3180:GOTO 1390
880 ON QQ GOTO 1180,1200,1220,1240,1260,1280,1300,1320,1340,1360
890 REM END OF VQ DATA INPUT
900 REM START OF 3 COMPMT DATA INPUT
910 CLS:WIDTH 80:PRINT "TO CHANGE ANY PARAMETER FROM ITS DEFAULT (NORMAL) VALUE
ENTER THE NUMBER AND"

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920 PRINT :PRINT "PRESS <RETURN KEY>. TO START CALCULATIONS PRESS <RETURN KEY> W
ITHOUT ENTERING "
930 PRINT :PRINT "A NUMBER."
940 LOCATE 7,30:PRINT "NORMAL VALUES","PRESENT VALUES"
950 LOCATE 8,1:PRINT "1. TIDAL VOLUME (VT)          500 ML",,VT
960 PRINT "2. TOTAL DEAD SPACE (VD)          150 ML",,VD
970 PRINT "3. RESPIRATORY FREQUENCY (F)      15 / MIN.",,F
980 PRINT "4. INSPIRED O2 (FIO2)            .21",,FIO2
990 PRINT "5. O2 CONSUMPTION (VO2)          300 ml/MIN",,VO2
1000 PRINT "6. RQ (VCO2/VO2)                .8",,R
1010 PRINT "7. PULM.BLD.VOL.(PBV)           500 ml",,PBV
1020 PRINT "8. CARDIAC OUTPUT (QT)           5.5 L/MIN.",,QT
1030 PRINT "9. DIFFUSION O2(DLO2)            32 ML/MIN/mm Hg",DLO2
1040 PRINT "10. SHUNT FLOW (QS)              .25 L/MIN.",,QS
1050 PRINT "11. HEMOGLOBIN (HGB)            15 GM/DL",,HGB
1060 PRINT "12. BASE EXCESS                  0 MEQ/L",,BE
1070 PRINT "13. TEMPERATURE (TEMP)          37 DEG C",,TEMP
1080 LOCATE 24,1:PRINT "
";
1085 IF R>1 OR R<.7 OR TEMP>42 OR TEMP<32 OR ABS(BE)>15 OR HGB>22 GOTO 3260
1090 LOCATE 24,1:INPUT;QQ
1100 IF QQ=0 THEN GOSUB 1660:GOSUB 1500:GOSUB 3180:GOTO 1390
1110 ON QQ GOTO 1120,1140,1160,1180,1200,1220,1240,1260,1280,1300,1320,1340,1360

1120 LOCATE 24,1:INPUT ;"ENTER NEW VALUE FOR TIDAL VOLUME.";VT:LOCATE 8,57:PRINT
"      ":LOCATE 8,57:PRINT VT:IF Q3=2 GOTO 850:REM Q3 DIRECTS PROGRAM BACK TO VQ
SUBROUTINE
1130 GOTO 1080
1140 LOCATE 24,1:INPUT ;"ENTER NEW VALUE FOR DEAD SPACE.";VD:LOCATE 9,57:PRINT "
":LOCATE 9,57:PRINT VD:IF Q3=2 GOTO 850
1150 GOTO 1080
1160 LOCATE 24,1:INPUT ;"ENTER NEW VALUE FOR RESPIRATORY RATE.";F:LOCATE 10,57:P
RINT "      ":LOCATE 10,57:PRINT F:IF Q3=2 GOTO 850
1170 GOTO 1080
1180 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR FIO2.";FIO2:LOCATE 11,57:PRINT "
":LOCATE 11,57:PRINT FIO2:IF FIO2>3 THEN CLS:PRINT "PATIENT HAS DEVELOPED CONVUL
SIONS DUE TO CNS O2 TOXICITY.":GOTO 1390
1185 IF Q3=2 GOTO 850
1190 GOTO 1080
1200 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR VO2.";VO2:LOCATE 12,57:PRINT "      ":
LOCATE 12,57:PRINT VO2:IF Q3=2 GOTO 850
1210 GOTO 1080
1220 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR RQ.";R:VCO2=R*VO2:LOCATE 13,57:PRINT
"      ":LOCATE 13,57:PRINT R:IF Q3=2 GOTO 850
1230 GOTO 1080
1240 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR PBV.";PBV:LOCATE 14,57:PRINT "      ":
LOCATE 14,57:PRINT PBV:IF Q3=2 GOTO 850
1250 GOTO 1080
1260 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR QT.";QT:LOCATE 15,57:PRINT "      ":LO
CATE 15,57:PRINT QT:IF Q3=2 GOTO 850
1270 GOTO 1080
1280 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR DLO2.";DLO2:LOCATE 16,57:PRINT "
":LOCATE 16,57:PRINT DLO2:IF Q3=2 GOTO 850
1290 GOTO 1080
1300 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR QS.";QS:LOCATE 17,57:PRINT "      ":LO
CATE 17,57:PRINT QS:IF Q3=2 GOTO 850
1310 GOTO 1080
1320 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR HGB.";HGB:LOCATE 18,57:PRINT "      ":
LOCATE 18,57:PRINT HGB:IF Q3=2 GOTO 850
1330 GOTO 1080
1340 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR BASE EXCESS";BE:LOCATE 19,57:PRINT "
":LOCATE 19,57:PRINT BE:IF Q3=2 GOTO 850

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1350 GOTO 1080
1360 LOCATE 24,1:INPUT;"ENTER NEW VALUE FOR TEMP.";TEMP:LOCATE 20,57:PRINT "
":LOCATE 20,57:PRINT TEMP:IF Q3=2 GOTO 850
1370 GOTO 1080
1380 REM END OF DATA INPUT
1390 WIDTH 80:PRINT :PRINT "WHAT DO YOU WANT TO DO NOW?"
1400 PRINT "    1. COMPARE PRESENT BLOOD GAS DATA WITH NEW."
1410 PRINT "    2. CALCULATE ANOTHER SET OF BLOOD GAS DATA."
1420 PRINT "    3. EXAMINE O2 DELIVERY AT TISSUE LEVEL"
1430 PRINT "    4. EXIT PROGRAM"
1440 PRINT :INPUT "ENTER NUMBER OF CHOICE THEN PRESS RETURN KEY.":Q1
1450 IF Q1=1 THEN QSUM=0:TOTO2=0:CLS:GOTO 440
1460 IF Q1=2 THEN CLS:Q1=0:GOTO 440
1470 IF Q1= 4 THEN CLS:END
1480 IF Q1=3 THEN NTOT=0:TOTO2=0:SW=0:SHIFT=AShift:GOTO 2320
1490 IF Q1<1 OR Q1>4 THEN CLS:PRINT"THIS IS NOT A CHOICE TRY AGAIN.":GOTO 1390
1500 CLS:SCREEN 1,0:REM FICK GRAPHICS DRAWING
1510 LINE(50,5)-(50,165),2:LINE-(250,165),2:LINE(50,5)-(48,5),2:LINE(50,45)-(48,
45),2:LINE(50,85)-(48,85),2:LINE(50,125)-(48,125),2:LINE(50,165)-(48,165),2:LINE
(50,165)-(50,167),2:LINE(89,165)-(89,167),2:LINE(131,165)-(131,167),2
1520 LINE(171,165)-(171,167),2
1530 LINE(211,165)-(211,167),2:LINE(251,165)-(251,167),2:LOCATE 1,5:PRINT"20":LO
CATE 6,5:PRINT "15":LOCATE 11,5:PRINT "10":LOCATE 16,6:PRINT "5":LOCATE 22,6:PRI
NT "0":LOCATE 22,12:PRINT "5":LOCATE 22,17:PRINT "10":LOCATE 22,22:PRINT "15"
1540 LOCATE 22,27:PRINT "20";
1550 LOCATE 10,2:PRINT "CV02":LOCATE 13,1:PRINT "(VOL%)":LOCATE 23,14:PRINT "CAO
2 (VOL%)"
1560 LINE(112,143)-(239,16),1:LINE(133,143)-(260,16),1:LINE(155,143)-(282,16),1
1570 LOCATE 1,26:PRINT "FR=20 16 12"
1580 IF V02=300 THEN LOCATE 2,28:PRINT "Q= 6 4 3"
1590 IF Q1=0 THEN X1=50+CA02*8:Y1=165-(CV02*8):CIRCLE(X1,Y1),4
1600 IF Q1=1 THEN X2=50+CA02*8:Y2=165-(CV02*8):LINE(X2-2,Y2-2)-(X2+2,Y2+2),,B:CI
RCLE(X1,Y1),4:LINE(X1,Y1)-(X2,Y2)
1610 LOCATE 2,10:PRINT "INITIAL VALUES":LOCATE 3,11:PRINT "PaO2 ="INT(P102):LOCA
TE 4,11:PRINT "PaCO2="INT(P1CO2):LOCATE 5,11:PRINT "pH  ="INT(100*APH1)/100:LOC
ATE 6,11:PRINT "HCO3 ="INT(HCO31):LOCATE 7,11:PRINT "PV02 ="INT(P1V02)
1620 LOCATE 8,8:PRINT "ALV.VENT="VA1
1630 IF Q1=1 THEN LOCATE 14,28:PRINT "FINAL VALUES":LOCATE 15,29:PRINT "PaO2 ="I
NT(PA02):LOCATE 16,29:PRINT "PaCO2="INT(PACO2):LOCATE 17,29:PRINT "pH  ="INT(AP
H*100)/100:LOCATE 18,29:PRINT "HCO3 ="INT(HCO3):LOCATE 19,29:PRINT "PV02 ="INT(P
V02)
1640 IF Q1=1 THEN LOCATE 20,25:PRINT "ALV.VENT="INT(VA)
1650 RETURN:END OF FICK GRAPHIC
1660 CLS:REM START OF PULMONARY CAP. CALCULATION SUBROUTINE
1670 LOCATE 24,1:PRINT : "CALCULATIONS IN PROGRESS;BE PATIENT ";
1680 IF Q3=2 THEN LOCATE 5,1:PRINT "VQ RATIO","PA02","PAC02","O2 UPTAKE","CUM.VA
"
1690 CV02=HGB*1.34*.95*(QT-QS)/QT-V02/(10*QT):IF CV02<2 THEN CLS:PRINT "PATIENT
HAS DIED DUE TO SEVERE HYPOXIA.":GOTO 1390:REM INITIAL APPROXIMATION OF VENOUS O
2 CONTENT
1700 VA=(VT-VD)*F
1710 AV02=V02/(QT*10)
1720 TIME=PBV*60*.15/(QT*1000):O2CAP=HGB*1.34
1730 P102=713*F102
1740 MODE=1:TOTO2=0 'RESET FOR NEXT RUN
1750 IF Q3=1 THEN VQ=VA/((QT-QS)*1000)
1760 IF Q3=2 THEN VA=0
1770 IF Q3=2 THEN FOR MODE=1 TO N:REM Q3=2 LINES USED IN VQ CALCULATIONS
1780 IF Q3=2 THEN LOCATE 3,10:PRINT " MODE="MODE
1790 IF Q3=2 THEN FOR PROBIT=-3 TO 3
1800 IF Q3=2 THEN VQ=EXP(LOG(VQ(MODE))+STDV(MODE)*PROBIT):IF VQ<.05 THEN VQ=.05:
REM PREVENTS OVERFLOW CONDITIONS
1810 IF PROBIT=-3 THEN FRACT=.005

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1820 IF PROBIT=-2 THEN FRACT=.05
1830 IF PROBIT=-1 THEN FRACT=.24
1840 IF PROBIT=0 THEN FRACT=.4
1850 IF PROBIT=1 THEN FRACT=.24
1860 IF PROBIT=2 THEN FRACT=.05
1870 IF PROBIT=3 THEN FRACT=.005
1880 FRACT=FRACT*QF(MODE) 'CONVERTS TO ACTUAL QT FRACTION
1890 IF Q3=2 THEN PACO2=.863*VCO2/(VQ(MODE)*QT) ELSE PACO2=VCO2*863/VA:IF PACO2>
100 THEN CLS:PRINT "PATIENT HAS DIED OF ASPHYXIA.":GOTO 1390
1900 PVC02=PACO2+VCO2/(QT*7.5):REM PVC02 SETS MAX. PALVCO2
1910 PCAPCO2=R*AVO2*10/VQ*(FIO2+(1-FIO2)/R):IF PCAPCO2>PVC02 THEN PCAPCO2=PVC02
1920 PCO2=PCAPCO2:IF PCO2<1 THEN PCO2=1
1930 RATIO=PCAPCO2/40:IF RATIO<.1 THEN RATIO=.1:REM SETS PHYSIOL. LIMIT
1940 HCO3=24.4+HGB*.6*LOG(RATIO)+BE:IF HCO3<4 THEN HCO3=4
1950 CAPPH=6.1+(LOG(HCO3/((.029*PCAPCO2))/LOG(10))):PH=CAPPH
1960 CCAPO2=CV02 'PULM.CAP. O2 CONTENT
1970 O2CONT=CCAPO2:GOSUB 3190:PCAP02=PO2
1980 PALV02=PIO2-R*AVO2*10/VQ*(FIO2+(1-FIO2)/R)
1990 IF PALV02<PCAP02 THEN PALV02=PCAP02
2000 IF Q3=1 THEN VQ=VA/((QT-QS)*1000)
2010 LOCATE 1,37:PRINT " "
2020 FOR T=0 TO TIME STEP TIME/50:REM START OF DIFFUSION CALCULATION
2030 DV02=(PALV02-PCAP02)*DLQ2*TIME/(50*60)
2040 CCAPO2=CCAPO2+DV02*100/(PBV*.2):REM CORRECTS FOR DL AND PULM CAP BL VOL.
2050 LOCATE 1,1:PRINT "PULM.CAP. O2 CONTENT FOR VQ SEGMENT "INT(100*VQ)/100="IN
T(10*CCAPO2)/10
2060 IF CCAPO2=>O2CAP THEN CCAPO2=O2CAP:PCAP02=PALV02:GOTO 2100
2070 O2CONT=CCAPO2:GOSUB 3190:PCAP02=PO2
2080 IF PCAP02=>PALV02 THEN PCAP02=PALV02:GOTO 2100
2090 NEXT T:REM END OF DIFFUSION CALCULATION
2100 UPTAKE=(CCAPO2-CV02)*FRACT:TOT02=TOT02+UPTAKE
2110 IF Q3=2 THEN VA=VA+VQ*(QT-QS)*FRACT*1000
2120 IF Q3=2 THEN LOCATE 3+MODE*7+PROBIT,1:PRINT INT(100*VQ)/100,INT(PALV02),INT
(PCAPCO2),INT(10*UPTAKE)/10,INT(VA)
2130 IF Q3=2 THEN NEXT PROBIT
2140 IF Q3=2 THEN CPV02=CV02+TOT02 'O2 CONTENT OF PULM. VEIN
2150 IF Q3=2 THEN NEXT MODE 'END OF PULMONARY CAP. CALCULATIONS
2160 LOCATE 24,1:PRINT "
";
2170 GOSUB 3180
2180 REM START OF ARTERIAL BLOOD GAS CALCULATIONS
2190 IF Q3=2 THEN CAO2=(QT-QS)/QT*CPV02+QS/QT*CV02 ELSE CAO2=(QT-QS)/QT*CCAPO2+
QS/QT*CV02
2200 HCO3=24.4+HGB*.6*LOG(PACO2/40)+BE
2210 APH=6.1+(LOG(HCO3/((.029*PACO2))/LOG(10))):PH=APH
2220 CAO2=CAO2+PCAP02*.0025 'ADDS DISSOLVED O2
2230 IF CAO2>O2CAP THEN PAO2=(CAO2-O2CAP)/.0025:GOTO 2250 'PO2 CALCULATED FROM D
ISSOLVED O2
2240 O2CONT=CAO2:GOSUB 3190:PAO2=PO2
2250 CV02=CAO2-V02/(QT*10):O2CONT=CV02
2260 VHC03=24.4+HGB*.6*LOG(PVC02/40)+BE:PCO2=PVC02
2270 VPH=6.1+(LOG(VHC03/((.029*PVC02))/LOG(10))):PH=VPH
2280 GOSUB 3190:PV02=PO2
2290 IF PV02<15 THEN LOCATE 10,1:PRINT "PATIENT HAS DIED DUE TO SEVERE HYPOXIA."
:GOTO 1390
2300 IF Q1=0 THEN PIO2=PAO2:PICO2=PACO2:APH1=APH:HCO31=HCO3:P1V02=PV02:VA1=INT(V
A):REM DATA STORED FOR COMPARISON
2310 RETURN 'END OF BLOOD GAS CALCULATIONS
2320 CLS 'TISSUE O2 SUBROUTINE
2330 RC=.0004:L=.075:K=2.1E-08:REM CAP&TISSUE CONSTANTS
2340 PCAP102=PAO2:CCAP102=CAO2
2350 INPUT "ENTER O2 CONSUMPTION PER 100 GM TISSUE PER MINUTE.":V02:V02=V02/100
2360 INPUT "ENTER BLOOD FLOW PER 100 GM TISSUE PER MINUTE.":QTT:Q=QTT/100

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2370 INPUT "ENTER PERCENT OF BLOOD FLOW SHUNTED BETWEEN ARTERY AND VEIN.";QS:MQ=
Q-QS*Q/100
2380 INPUT "ENTER CAP. DENSITY (# CAPS./CC TISSUE.";NMAX
2390 CVO2=CAO2-V02*100/Q:IF CVO2<.001 THEN CVO2=.001:PVO2=.001:REM PREVENTS DIVI
SION BY 0
2400 CLS:LOCATE 1,5:PRINT "CALCULATIONS IN PROGRESS":PRINT "PLEASE BE PATIENT."
2410 LOCATE 5,1:PRINT "THE PO2 IN THE TISSUE IS A COMPLEX FUNCTION OF MANY VARIA
BLES WHICH INCLUDE THE":PRINT :PRINT "RATE OF O2 CONSUMPTION, THE DENSITY AND LO
CATION OF MITOCHONDRIA, THE ARTERIAL"
2420 PRINT :PRINT "PO2 AND HEMATOCRIT, THE DIRECTION AND MAGNITUDE OF BLOOD FLOW
, THE "
2430 PRINT:PRINT "DISTANCE DOWN THE CAPILLARY AND THE INTERCAPILLARY DISTANCE AM
ONG OTHER":PRINT :PRINT "FACTORS. THE FOLLOWING SCREEN WILL SHOW A VERY SIMPLIFI
ED SCHEMATIC OF A THREE"
2440 PRINT :PRINT "DIMENSIONAL PLOT OF CAPILLARY LENGTH, INTERCAPILLARY DISTANCE
AND PO2 WHICH":PRINT :PRINT "WERE CALCULATED USING THE AVAILABLE DATA."
2450 GOSUB 2780:GOSUB 2550:GOSUB 2640:GOSUB 3180
2460 CLS:PRINT "VENOUS O2 CONTENT IS "INT(CVO2*10)/10
2470 PRINT :PRINT "VENOUS PO2 IS "PVO2
2480 PRINT :PRINT "OXYGENATED TISSUE="INT(TOTO2/V02*100)%"
2490 PRINT :PRINT "% CAPS. USED="INT(NTOT/NMAX*100)
2500 PRINT :PRINT "MEAN TISSUE PO2 (KROGH MODEL)="INT(TOTPO2/SUM)
2510 PRINT :PRINT "MINIMAL PO2 (PIIPER MODEL)="PTMIN
2520 PRINT "MAXIMAL TISSUE PO2 (PIIPER MODEL)="PTMAX
2530 PRINT :PRINT "DIFFUSION/PERFUSION RATIO VARIES FROM "DQMIN" TO "DQMAX
2540 GOSUB 3180:GOTO 1390
2550 'CAPILLARY GRAPHIC
2560 CLS:SCREEN 1,0
2570 CIRCLE (50,13),7,1:CIRCLE(200,13),7,1
2580 LOCATE 1,6:PRINT "CAP.";:LOCATE 1,25:PRINT "CAP.";:LOCATE 1,15:PRINT INT(RT
(1)*10000);
2590 LINE(193,13)-(57,13),1:LINE(50,20)-(50,120),1:LINE-(165,175),1:LINE-(305,17
5),1:LINE(165,175)-(165,175-PVO2*100/PAO2),1:LINE(305,175)-(305,175-PVO2*100/PAO
2),1
2600 LOCATE 10,4:PRINT "PO2";:LOCATE 16,6:PRINT "O";:LOCATE 24,24:PRINT "DISTANC
E (MICRA)";:LOCATE 23,29:PRINT INT(RT(1C)*10000);
2610 LOCATE 23,21:PRINT "O";:LOCATE 3,1:PRINT "a="PAO2:Y%=22-PVO2/PAO2*13:LOCATE
Y%-2,37:PRINT "Pv02";:LOCATE Y%,37:PRINT PVO2;:LOCATE 23,39:PRINT "O";
2620 LOCATE 17,8:PRINT "L":LOCATE 18,10:PRINT "E":LOCATE 19,12:PRINT "N":LOCATE
20,14:PRINT "G":LOCATE 21,16:PRINT "T":LOCATE 22,18:PRINT "H";
2630 RETURN
2640 'PLOTING SUBROUTINE
2650 XA=57:YA=13:XB=193:YB=13
2660 FOR L%=1 TO 10
2670 FOR D%=1 TO 20
2680 X%=50+L%*.5*20+D%*7.5:Y%=20+.5*L%*10+(100-PTO2(L%,D%)*100/PAO2):IF Y%>169 T
HEN Y%=169
2690 IF D%=1 THEN LINE(X%,Y%)-(XA,YA),3:XA=X%:YA=Y%
2700 IF PTO2(L%,D%)=0 THEN PSET(X%,Y%),0:H=H+1:GOTO 2720:REM FOR A=-10 TO 10:LIN
E(X%+A,Y%)-(X%+A,169),0:NEXT:GOTO 2030
2710 PSET(X%,Y%),2
2720 IF D%=20 THEN LINE(XB,YB)-(X%,Y%),3:XB=X%:YB=Y%
2730 SUM=SUM+1:TOTPO2=TOTPO2+PTO2(L%,D%)
2740 NEXT
2750 NEXT
2760 LINE(XA,YA)-(165,175-PVO2/PAO2*100):LINE(XB,YB)-(305,175-PVO2/PAO2*100)
2770 RETURN
2780 REM CALCULATION SUBROUTINE
2790 L%=0:CCAP1(L%)=CAO2:PCAP1(L%)=PAO2:PCAP2(L%)=PCAP1(L%)
2800 N=NMAX/5
2810 LOCATE 19,1:PRINT "CAPILLARY DENSITY BEING CALCULATED"
2820 FOR L%=1 TO 10
2830 IF N>NMAX THEN N=NMAX:SW=1

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2840 RT(L%)=SQRT(1/(N*L*4)) 'KROGH CYLINDER RADIUS
2850 CCAP1(L%)=CCAP1(L%-1)-VO2*10/MQ:IF CCAP1(L%)<.01 THEN CCAP1(L%)=0:PCAP1(L%)
=0:PCAP2(L%)=0:GOTO 2920
2855 IF CAO2>O2CAP THEN PAO2=(CAO2-O2CAP)/.0025:GOTO 2870
2860 O2CONT=CCAP1(L%):GOSUB 3190:PCAP1(L%)=PO2:PCAP2(L%)=PO2
2870 AREA=3.1416*2*RC*N*L/10
2880 DPCO2=(VO2*R/(MQ*7.5))/10:PCAPCO2=PACO2+DPCO2*L%:PCO2=PCAPCO2
2890 CAPHCO3=24.4+HGB*.6*LOG(PCAPCO2/40)+BE
2900 CAPPH=6.1+(LOG(HCO3/(.029*PCAPCO2))/LOG(10)):PH=CAPPH
2910 GOSUB 3190:PCAP1(L%)=PO2
2920 D1O2(L%)=PCAP1(L%)*AREA*K/RT(L%)
2930 IF SW=1 GOTO 2960
2940 IF D1O2(L%)<VO2/10 THEN N=N*1.02:GOTO 2830:REM DETERMINES# OF CAPS.
2950 IF D1O2(L%)>1.02*VO2/10 THEN N=N*.97:GOTO 2830:REM " " "
2960 NTOT=NTOT+N/10:TOTO2=D1O2(L%)+TOTO2
2970 LOCATE 20,2*L%:PRINT L%
2980 NEXT
2990 LOCATE 19,1:PRINT "TISSUE O2 TENSION BEING CALCULATED"
3000 O2CONT=CV02:IF O2CONT<0 THEN O2CONT=0
3005 GOSUB 3190:PVO2=PO2:IF PVO2<0 THEN PVO2=0
3010 FOR L%=1 TO 10
3020 FOR D%=1 TO 10
3030 X=D%*RT(L%)/10
3040 PTO2(L%,D%)=PCAP1(L%)-VO2*RCΔ2/(4*K)*(2*(RT(L%)/RC)Δ2*LOG(X/RC)-((X/RC)Δ2-1
)):IF PTO2(L%,D%)<0 THEN PTO2(L%,D%)=0
3050 PTO2(L%,21-D%)=PTO2(L%,D%)
3060 LOCATE 21,1:PRINT "%LENGTH=";L%*10;
3070 LOCATE 22,1:PRINT "O2 DELIVERED/O2 CONSUMED="INT(100*TOTO2/VO2)/100
3080 LOCATE 23,1:PRINT "% CAPS USED=" INT(NTOT/NMAX*100)
3090 NEXT
3100 NEXT
3110 AREA=RC*2*3.1416*L*NTOT
3120 QBB=MQ*(CAO2-CVO2)/((PAO2-PVO2)*100):DQ1=.005*2*3.1416*3*K/.005/QBB
3130 DQMIN=INT(100*AREA*K/RT(1)/QBB)/100:REM DIFF/PERF COEFF
3140 DQMAX=INT(100*AREA*K/RT(10)/QBB)/100:REM " "
3150 PTMIN=PAO2+(PVO2-PAO2)/(1-EXP(-DQMIN)+DQ1):IF PTMIN<0 THEN PTMIN=0
3160 PTMAX=PAO2+(PVO2-PAO2)/(1-EXP(-DQMAX)+DQ1):IF PTMAX<0 THEN PTMAX=0
3170 RETURN
3180 LOCATE 25,1:INPUT "PRESS RETURN KEY TO CONTINUE";QQ:CLS:RETURN
3190 REM START OF HGB SATURATION CORRECTION
3200 X=EXP(LOG((100.0006*O2CAP/(O2CONT*100)-1)/11934.6)/-2.733796)-4.81
3210 A=.024*(37-TEMP)
3220 B=.2*(PH-7.4)
3230 C=.06*(LOG(40)-LOG(PCO2))/LOG(10)
3240 PO2=X/10Δ(A+B+C)
3250 RETURN 'END OF HGB DISSOC CURVE CORRECTION

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